Target lesion characteristics in failing vein grafts predict the success of endovascular and open revision

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Objectives: **This study examined the association of anatomic and temporal characteristics of graft-threatening lesions with the efficacy of percutaneous and open graft revision for failing infrainguinal vein grafts.**

Methods: **Consecutive open and endovascular revisions for graft threatening lesions were reviewed. We evaluated graft durability and individual target lesion response to open and endovascular treatment to determine characteristics that may influence outcomes. Treatment failure was defined as target lesion restenosis or graft occlusion.**

Results: **Eighty-four (58 endovascular, 26 open) infrainguinal vein graft revisions were performed in 67 failing, nonthrombosed infrainguinal vein grafts. Primary assisted graft patency at 5 years was 63% (95% confidence interval [CI], 46% to 77%). Follow-up was 29.5 ± 19.2 months. Grafts treated for early lesions (<6 months) failed (occlusion or need for additional interventions) more frequently than those with late occurring lesions ($P = .03$). Overall target lesion revascularization patency was 45% (95% CI, 32% to 58%) at 3 years. Average time to target lesion revascularization failure was 7.5 months, with no significant difference noted between endovascular and open treatment groups. Overall target lesion revascularization patency at 3 years was also not significantly different between open and endovascular groups at 54% (95% CI, 30% to 73%) vs 41% (95% CI, 25% to 56%; $P = .15$). When divided by early and late-occurring target lesions, endovascular treatment of early lesions was associated with inferior patency compared with open procedures; no difference in patency was seen between treatment groups for late lesions. When divided by target lesion location (anastomotic vs mid-graft), treatment for both proximal and distal anastomotic target lesion was associated with inferior patency compared with mid-graft revision at 32% (95% CI, 17% to 47%) vs 62% (95% CI, 37% to 87%) at 3 years ($P = .03$). In addition, although results of anastomotic target lesion treatment significantly favored open repair, even open repair of anastomotic target lesions was associated with a <50% patency rate at 3 years. In contrast, mid-graft target lesions treated with open revisions were uniformly successful compared with a 54% patency at 3 years with endovascular treatment ($P = .24$). Short lesions (<2 cm) fared equally well with either endovascular or open treatment. Univariate analysis noted only anastomotic treatment was associated with significantly increased odds of failure.**

Conclusion: **Grafts that develop early lesions fare poorly regardless of treatment modality. Lesions involving anastomoses of failing grafts are better treated with open revision, but patency after treatment of such lesions is still worse than treatment of mid-graft lesions. In contrast, the method of treatment does not influence outcome after treatment of mid-graft target lesions. Thus, endovascular therapy should be reserved for focal, late-appearing lesions involving the mid-graft. (J Vasc Surg 2007;46:1167-72.)**

Determining the utility of any treatment for failing grafts is made difficult by the variety of treatment options, the frequent multiplicity of lesions, and specific lesion characteristics, including length, location, and temporal development after graft implantation. We hypothesize that graft-threatening lesions have specific characteristics that affect the efficacy of percutaneous and open graft revision for failing infrainguinal vein grafts and propose a rational treatment regime for preservation of graft patency.

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Competition of interest: none.

Presented at the Thirty-first Annual Meeting of the Southern Association for Vascular Surgery, Rio Grande, Puerto Rico, Jan 17-20, 2007.

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CME article

0741-5214/$32.00

doi:10.1016/j.jvs.2007.08.017

METHODS

All consecutive open and endovascular revisions for graft-threatening lesions performed between January 2000 and August 2006 in autogenous infrainguinal bypass grafts were reviewed. The study was approved by the Institutional Review Board at the University of Texas Health Science Center at San Antonio. Only repairs of lesions considered part of the graft were included, with deliberate exclusion of any repairs for native inflow or outflow arterial lesions.

Failing grafts were identified through a routine graft surveillance protocol using duplex ultrasound imaging in a single-center noninvasive vascular laboratory accredited by the Intersocietal Commission for the Accreditation of Vascular Laboratories. Duplex diagnostic criteria for lesions requiring interventions were used from previously described parameters.[1,2] Lesions with peak systolic velocity >300 cm/s, mid-graft velocities <45 cm/s, and velocity ratios >3.5 were considered critical in nature, prompting revision. Methods of repair were not randomized and var-

ied according to the discretion of the operating surgeon/interventionalist.

Graft-specific data collection. Patient characteristics, demographics, and cardiovascular risk factors were recorded. In addition, specific characteristics related to the original graft procedure were delineated, including operative indications, conduit type and configuration, inflow and outflow source, and duplex ultrasound scan results that detected the failing grafts. The number of lesions per graft and time to treatment of the first lesion were recorded. Grafts had to be patent at the time of revision, and grafts subjected to thrombolytic therapy or thrombectomy before revision were not included. With the full understanding that all the grafts studied had lost primary patency, reporting standards established by the Society for Vascular Surgery/International Society for Cardiovascular Surgery were used. Graft survival was calculated from the time of the original procedure. Freedom from all-cause graft failure inclusive of lesion recurrence and graft occlusion defined primary patency. Freedom from graft occlusion, regardless of the need for additional intervention to maintain patency, was defined as primary assisted patency.

Target lesion-specific data collection. Locations of lesions were classified in two main groups: juxtaanastomotic (<2 cm of an anastomosis), or intrinsic vein graft (mid-graft), arbitrarily defined as the area between the 2 cm range of either anastomosis. In addition, data specific to the target lesions were collected, including lesion length (short, ≤2 cm; long, >2 cm) and temporal development after original graft implantation (early, ≤6 months; late, >6 months). The revision method was recorded per target lesion, defined as target lesion revascularization (TLR), and was broadly categorized into open surgical and endovascular techniques, Target lesion end points were based on the initial method of treatment per target lesion.

TLR failure occurred if (1) significant target lesion restenosis occurred, (2) the initial treatment failed (intent-to-treat), or (3) the graft occluded, regardless of the presumed culprit lesion or cause. Therefore TLR primary patency was defined by the combined end point of freedom from reintervention after target lesion revascularization and freedom from graft occlusion. Significant restenosis was defined by the same diagnostic duplex criteria for the primary graft-threatening lesion as detailed. Duration of TLR patency was recorded from the date of the first TLR (t = 0) to the date of failure (t = date of event) and should not be confused with graft-specific survival end points, which begin with original graft implantation.

Statistical analysis. Statistical analyses were performed using SAS 9.1 software (SAS Inc, Cary, NC). Continuous variables were expressed as mean ± standard deviation and categoric data as percentages. Analysis of binary outcomes and categoric variables was performed using the Fisher exact test. Logistic regression was used to calculate odds ratios) with 95% confidence intervals (CI). Continuous variables were compared with the two-sampled *t* test with equal variances.

Table I. Original procedure characteristics

Characteristics	*No.*	*%*
Indication		
Claudication	10	15
Limb threat	57	85
Inflow source		
Femoral artery		
Common	42	63
Deep	4	6
Superficial	16	24
Popliteal artery	5	7
Conduit		
Reversed	37	55
Nonreversed	18	27
Alternative	12	18
Outflow level		
Popliteal artery		
Above knee	11	16.4
Below knee	21	31.3
Tibial artery	31	46.3
Pedal artery	4	6.0

Survival data, including primary graft patency, assisted primary graft patency, and TLR primary patency, were determined using Kaplan-Meier life table analysis. Survival results were compared with the log-rank test for equality of survival functions. A value of $P < .05$ was considered significant for all analyses. However, when two survival probabilities at specific time points were compared, the *Z* test with Bonferroni correction was used to determine significance to ensure that the chance of false interference was at most 5%.

RESULTS

During the study period, 64 patients (51 men, 13 women; mean age, 63 ± 9.9 years) with 67 lower extremity autogenous infrainguinal bypasses underwent revision for graft threatening lesions. Indications and operative details for the original bypass procedures, including graft configuration, type of conduit, and proximal and distal anastomotic levels of the original lower extremity bypasses, are summarized in Table I. Preintervention data from duplex imaging was available for 63 grafts (97%). Of those grafts, 35 (56%) had low mid-graft velocities (<45 cm/s) with or without discrete stenoses. The remaining 28 grafts had discrete stenoses without associated low mid-graft velocities.

The initial intervention for graft-threatening lesions was performed a mean of 12 ± 12 months after the original procedure. Thirty-three grafts (49.3%) were treated for index graft lesions that developed ≤6 months after original graft procedure, and the other 34 grafts (50.7%) developed lesions >11 months after original bypass. Multiple lesions developed in 18 of 67 grafts (26.9%), and 84 lesions were repaired. Twenty-seven of the repaired lesions (32%) occurred in the main graft body, and 57 (68%) occurred at an anastomosis: 38 (67%) were at the proximal anastomosis and 19 (33%) at a distal anastomosis. Table II summarizes the primary TLR methods.

Table II. Characteristics of primary target lesion revascularization methods

Primary TLR	*N*	*%*
Open surgical	26	31
Vein patch angioplasty	19	
Interposition graft	7	
Endovascular	58	69
Conventional PTA	48	
Cutting balloon PTA	10	
Total	84	100

TLR, Target lesion revascularization; *PTA,* percutaneous transluminal angioplasty.

Table III. Complications by procedure type

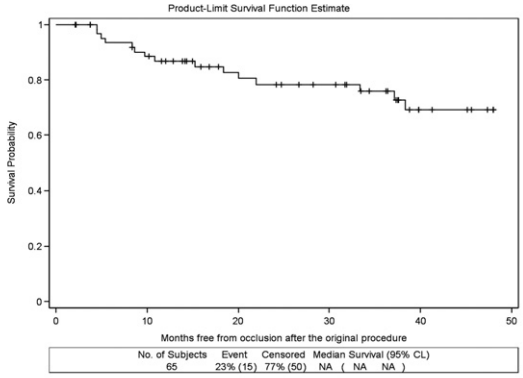
Complication	*No.*
Open procedures	
Infected lymphocele	1
Endovascular procedures	
Residual stenoses	4
Distal thromboembolism	2
Access site	
False aneurysm	3
Infection	2
Hematoma	1
Total	13

Endovascular therapy was applied with equal frequency to proximal anastomotic (34.5%), mid-graft (34.5%), and distal anastomotic lesions (31%) compared with an uneven distribution in the open group of proximal anastomotic (69.2%), mid-graft (26.9%), and distal anastomotic (3.8%; $P = .003$) repairs. Complications occurred in 13 (15%) primary TLR procedures (12 endovascular, 1 open; Table III).

Graft-specific results. With a mean follow-up of 29.5 ± 19.2 months, 5-year primary assisted graft patency (freedom from graft occlusion) was 63% ± 8.0% (95% CI, 46% to 77%; Fig 1). Primary patency at 5-years (freedom from all-cause failures) was 29% ± 7.4% (95% CI, 16% to 44%; Fig 2). Primary assisted graft patency was not influenced by age, gender, or traditional atherosclerotic risk factors, nor by vein graft configuration, anastomotic location, or operative indication. Furthermore, time to onset of the index graft lesion, multiplicity of lesions, initial method of treatment for the first TLR (open vs endovascular), need for additional second TLR procedures for recurrent lesions involving the index site, and need for additional interventions for other lesions outside the index lesion did not have any significant influence on overall graft survival.

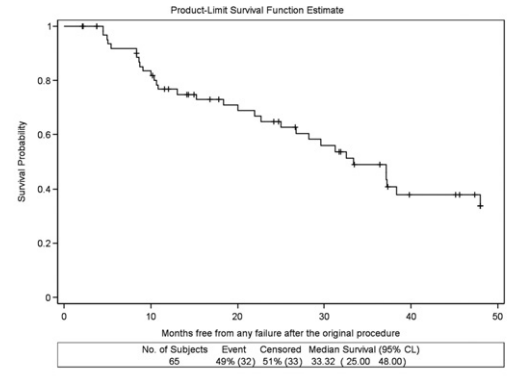
A trend was noted toward worse patency among grafts that developed early lesions (log rank, $P = .06$); and, separated by specific time intervals, grafts with early lesions had significantly worse patency at 12 and 24 months of 71% (95% CI, 51% to 84%) and 65% (95% CI, 42% to 80%) compared with grafts with late-appearing lesions at 100% and 90% (95% CI, 72% to 97%; $P < 0.001$ and $P = .026$, respectively). However, these differences in overall primary assisted graft patency were lost during the remainder of follow-up, with no difference seen between groups at 48 months: early lesion grafts were at 65% (95% CI, 42% to 80%) vs late lesion grafts at 75% (95% CI, 52% to 88%; $P = .45$).

Results for primary patency (freedom from all cause failure) were similar to primary assisted patency results, and only the development of early lesions significantly influenced the primary patency of a graft ($P = .03$). Finally, grafts with low mid-graft velocities (<45 cm/s) had significantly worse primary patency compared with grafts with normal mid-graft velocities and discrete stenoses ($P = .039$). However, these findings did not translate to primary assisted patency results, where no differences in rates of occlusion were noted between the two groups.



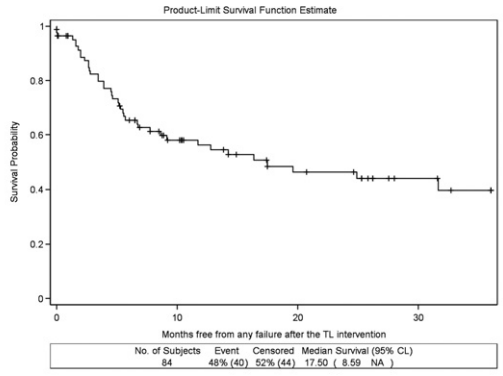
Months	0	3	6	12	24	36	48
Grafts at Risk	67	62	59	47	33	23	8

Fig 1. Overall graft survival expressed as primary assisted patency, which represents freedom from graft thrombosis regardless of additional revision. *CL,* Confidence limits.



Months	0	3	6	12	24	36	48
Grafts at Risk	67	63	55	42	28	16	6

Fig 2. Overall graft survival expressed as primary patency. This represents freedom from all-cause failures, including graft thrombosis, target lesion recurrence, and intervention for additional graft-threatening lesions. *CL,* Confidence limits.



Months	0	3	6	12	24	36
Lesions at Risk	84	69	53	31	20	10

Fig 3. Overall target lesion revascularization primary patency. This represents survival data specific to a particular target lesion intervention rather than overall graft survival. *CL,* Confidence limits.

Target lesion revascularization results. The 3-year TLR primary patency was 45.4% (95% CI, 32% to 58%; Fig 3), and the average time to TLR failure was 7.5 ± 8.4 months. When grouped by specific method of TLR (patch, interposition graft, or balloon angioplasty), no differences were seen in overall TLR primary patency ($P = .12$). When grouped according to TLR strategy (open vs endovascular), TLR primary patency was also not different over 3 years if a lesion was treated with open surgery or endovascular intervention: 52% (95% CI, 28% to 71%) vs 31% (95% CI, –12% to 51%; $P = .17$), respectively.

Additional secondary interventions were required to salvage primary TLR failures in 28 lesions (35%): 22 (73%) required a single secondary intervention, six (20%) required two additional procedures, and two (7%) required three repeat interventions. However, the mean number of additional primary target lesion reinterventions did not differ between lesions treated initially with endovascular therapy (1.3 ± 0.5) compared with lesions initially treated with surgical therapy (1.8 ± 0.8.). In contrast, TLR recurrence was significantly higher among patients who had complications compared with uncomplicated procedures ($P = .013$).

Treatment of shorter lesions (≤2-cm long) was associated with significantly better patency than treatment of longer lesions (Table V), and treatment of longer lesions was associated with uniformly poor primary patency regardless of treatment modality (Table IV). Although no difference was seen in treatment durability for early (≤6 months) lesions compared with lesions developing >6 months (Table V), early lesions treated with endovascular therapy also had inferior patency compared with open surgery at 3 years (Table IV). In contrast, this difference was not seen with treatment of late occurring lesions, with both treatment modalities being associated with similar 3-year patency rates.

Table IV. Target lesion primary patency at 3 years by type of repair

Lesion characteristic	*Open*	*Endovascular*	P
Short, ≤2 cm	65%	35%	.12
Long, >2 cm	22%	11%	.01
Early, ≤6 mon	64%	18%	.05
Late, >6 mon	43%	55%	.75
Anastomotic location	46%	16%	.01
Mid-graft location	100%	54%	.11

NS, Not significant.

Table V. Target lesion primary patency at 3 years

Lesion characteristic	*Overall*	P
Short, ≤2 cm	47%	.009
Long, >2 cm	13%	
Early, ≤6 mon	34%	.09
Late, >6 mon	50%	
Anastomotic location	32%	.03
Mid-graft location	62%	

NS, Not significant.

The comparison between anastomotic vs mid-graft target lesion location showed that patency for any interventions performed at an anastomotic site (proximal or distal) was inferior to that of interventions performed in the mid-graft (Table V). The 3-year patency of anastomotic stenoses treated with open repair was also superior to results of endovascular therapy of such lesions (Table IV). Furthermore, mid-graft lesions treated with open revisions were uniformly successful over 36 months compared with 54% TLR primary patency (95% CI, 28% to 74%) seen with endovascular therapy ($P = .24$). Finally, failure risk of TLR was 3-fold higher in the treatment of anastomotic lesions compared with mid-graft lesions by univariant analysis (odds ratio, 3.3; 95% CI, 1.2 to 8.7) whereas gender, risk factors, lesion length, lesion timing or initial TLR strategy (open vs endovascular) were not significant.

DISCUSSION

Our overall graft survival (primary assisted patency) of 63% at 5 years is consistent with other reports,[3-6] and the only characteristic that seemed to influence late graft patency was the development of lesions ≤6 months after the original bypass. This likely is a reflection of the aggressiveness of early occurring lesions and restenosis rates characteristic of treatment of these target lesions. Mills et al[2] suggested that most grafts that develop significant early lesions requiring revision have identifiable flow disturbances manifesting at implantation or ≤3 months after implantation.[2]

Therefore, it may be reasonable to assume that early lesions are indicative of poor initial conduit, a characteristic associated with compromised outcomes.[7,8]

Despite the unfavorable failure rate of maintenance procedures performed on grafts with early lesions, overall

late graft survival >48 months (primary assisted patency) for such grafts was not different compared with grafts undergoing similar procedures for later appearing lesions. This appears to be largely the result of aggressive intervention and reintervention on culprit lesions, further validating the need for maintenance intervention.

The response of a particular target lesion to intervention is the important focus of the current study. Treatment of shorter lesions was associated with significantly longer patency than treatment of longer lesions. Regardless of the modality of therapy, longer lesions were also associated with poor overall patency. However, open surgical treatment of long lesions, although ultimately resulting in failure, did take approximately 6 months longer to fail compared with endovascular therapy. Despite the uniform poor outcome for any revision of a long lesion, an open surgical strategy therefore appears to buy more time before the next failure event and may reduce the number of graft salvage procedures required to maintain overall primary assisted graft patency.

Endovascular treatment of early developing lesions has been discouraged.[3,5,9] Some postulate that the metabolic activity of these early lesions may predispose them to more rapid restenoses after intervention.[3,5] Our study results reaffirm these findings. Endovascular intervention was associated with worse outcomes for the treatment of early graft-threatening lesions, and open surgical intervention was associated with a much longer patency. This difference did not translate to the treatment of late lesions, however. With late lesions, there was no difference between the two treatment modalities: both were associated with similar failure rates. These observations indicate that equal success can be expected in treatment of late-onset lesions with open or endovascular methods, but greater consideration should be given to treat early lesions with open surgical techniques.

Perhaps the most interesting finding in our study was the effect of lesion location on revision outcomes. As noted by others, anastomotic lesions tend to respond less favorably to revision than lesions involving the main body of the vein graft.[10] In addition, although open repair of anastomotic lesions resulted in somewhat disappointing results, with approximately 50% of interventions failing during follow-up, endovascular treatment of anastomotic target lesions was even more disappointing.

The fate of interventions at anastomotic sites was also recently examined by Eagleton et al,[10] and contrary to our findings, they noted equivalent success rates between open surgical and endovascular treatment of these lesions, with 56% and 51% cumulative patency at 3 years between endovascular and open treatments, respectively. Despite the modest response associated with open surgical treatment for anastomotic lesions in our study, our endovascular results were worse. The inclusion of prosthetic grafts in the Eagleton et al study may explain the disparate results between our findings and their results.

Because performance of endovascular intervention applied to mid-graft lesions was better than anastomotic lesions, the argument for a more liberal application of percutaneous therapy to mid-graft lesions can be made. However, because open surgery was uniformly successful compared with a 54% patency after endovascular interventions, the use of percutaneous intervention should only be driven by an avoidance of a difficult surgical dissection, such as an anatomically tunneled graft in a large limb. That being said, our data note higher complication rates associated with percutaneous intervention compared with open intervention, and these complications were not insignificant and were associated with higher recurrence rates.

Our univariate analysis confirmed the results of the survival data in that the odds of successful primary target lesion revascularization were influenced mainly by anastomotic lesion location. Lesion length, lesion timing, and method of primary intervention were not significant contributors to the odds of failure. Moreover, despite the variability of target lesion behavior after intervention, it is important to remember that most target lesion failures take the form of recurrent lesions that can be secondarily treated before graft failure. Although the fate of a particular target lesion may be restenosis or recurrence, in the greater scheme of strategic graft health, the number of reinterventions bore no impact on ultimate graft survival. It can thus be concluded that persistence in treatment of graft-threatening lesions will result in durable occlusion free graft survival.

The generalizability of our findings is limited by the retrospective nature of this study because there may be other predictors of target lesion treatment success or failure that were not evaluated. The relatively small numbers in certain subgroups also make our data prone to type II error. In addition, the small sample size limits adequate exploration of outcomes related to the application of certain technologies such as cutting balloons or plaque excision devices. Also, the choice of intervention strategy was not random; thus, selection bias influenced the choice of treatment, and endovascular therapy was likely applied to anatomic situations that were considered more difficult to approach surgically. However, the study does provide a unique look at primary target lesion characteristics and behavior after different treatment strategies as well as the global impact on the more meaningful end point of graft survival.

CONCLUSION

Early lesions within failing lower extremity vein grafts appear to identify a compromised initial conduit. Such grafts fare poorly regardless of treatment modality and require extensive maintenance. Anastomotic lesions should be treated with open surgical methods, but clinicians should expect modest results. Long lesions and early lesions should also be preferentially treated with open repair, and endovascular therapy should be reserved for focal, late-appearing lesions involving the mid-graft.

AUTHOR CONTRIBUTIONS

Conception and design: RH, BT
Analysis and interpretation: RH, IJ, EC, BT
Data collection: RH, MS, RS, BT
Writing the article: RH, MS, IJ, BT

Critical revision of the article: EC, MS, BT
Final approval of the article: RH, BT
Statistical analysis: RH, IJ, EC, MS
Obtained funding: Not applicable
Overall responsibility: RH

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Submitted Feb 23, 2007; accepted Aug 6, 2007.

DISCUSSION

Dr Martin R. Back (Tampa, Fla). Duplex ultrasound surveillance after infrainguinal vein bypass construction has been associated with meaningful extension of vein graft longevity by allowing repair of stenotic lesions threatening patency. Graft revision methods have evolved over the last 10 years with increasing acceptance of percutaneous transluminal balloon angioplasty as a durable option. However, the optimal match of stenotic lesion type and treatment method has not been fully determined.

Dr Hagino and his colleagues at San Antonio have dissected their recent experience with secondary interventions for vein graft stenoses using a rigorous statistical analysis to identify factors prognosticating outcomes for endovascular and open techniques. Their findings are not dissimilar with other series, including our group's experience reported in 1999. Early developing, defined as occurring within 6 months of construction, and multiple lesions shortened graft patency regardless of treatment method. Interventions for anastomotic stenoses fared worse than repairs of graft body lesions. Stenoses longer than 2 cm and mid-graft lesions responded better to open repairs and probably accounted for a general trend towards a shorter interval to restenosis seen after endovascular interventions. Despite this latter trend, increased graft occlusion was not observed after endovascular repairs owing to the ability of serial scanning to effectively allow additional re-interventions for restenoses.

I would like to thank the authors for providing me the manuscript early and allowing me ample time to struggle through the complex data set and its interpretation. I will limit my comments regarding multiple problems with confusing patency definitions and text clarity that will mandate extensive revision and better explanation and illustration of their methods and results. I have several queries.

First, for completeness, what was your graft surveillance protocol pre- and post-interventions? Was intraprocedural duplex scanning used and/or how soon afterwards was imaging done as a gauge of procedural (or technical) success? What was your procedural end point for success after endovascular and open repairs, and did this have any bearing on later outcomes?

Second, vein graft diameter has been acknowledged as a prognostic factor for treatment durability after endovascular intervention with smaller diameter veins (less than 3.5 mm) behaving unfavorably. Have you recorded or could you extract graft diameter information from duplex scans prior to intervention to include this parameter in your statistical analysis?

Lastly, procedural-related complications were not detailed in the manuscript. Would their greater occurrence after open repairs influence risk/benefit considerations and affect selection of the preferred treatment method for any specific lesion characteristic?

Dr Ryan T. Hagino (San Antonio, TX). Thank you, Dr Back for your questions and comments. I have found the oftenquoted practice of defining endovascular clinical end points in terms of target lesion recurrence or reintervention rates unhelpful in clinical practice, and I really felt the more useful end point was overall graft patency, in other words, freedom from occlusion. And so we tried to present our data using cumulative assisted patency. In other words, we tried to distinguish between the end points of graft occlusion (primary assisted patency) vs the need for secondary interventions inclusive of graft occlusions (primary patency).

Regarding our graft surveillance protocol, certainly the South Florida group has championed this, and then we have adopted your guidelines for both revision and surveillance. In terms of intraprocedural duplex, for all open repairs—both reinterventions and the primary index procedure—we perform intraoperative duplex ultrasound. I did not include revisions that were performed at the time of surgery.

Following endovascular procedures, we have not been as aggressive as your group in terms of doing intraprocedural duplex imaging. It is something I would like to adopt, but the practicalities of our practice locations make intraprocedural scanning for endoluminal interventions a little bit more difficult than for open procedures. Therefore, we end up declaring technical success in relationship to residual stenosis rather than hemodynamic resolution of the offending lesion as would be seen with intraoperative duplex imaging, and I acknowledge that this leads to an inequality between the groups.

In terms of vein graft diameter, I do recognize that as a limitation and going back and trying to comb the data looking for graft specific diameter in the absence of a prospective protocol was impossible, so we were not able to follow that end point.

Regarding the procedure-related complications, certainly, open procedures have their fair share of wound complications. There was a significant difference in complications rates between the endovascular and the open groups, with the endovascular groups sustaining more complications. These complications were not insubstantial. I can recall at least two or three interventions where we had distal embolization and had to go after them with thrombectomy catheters and percutaneous extraction techniques, so it is by no means benign.